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# *The Health Costs of Motor-Vehicle-Related Air Pollution*

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**Abstract**

Motor vehicles have significantly larger health costs than previously reported. Particulates are the most damaging pollutant, while ozone and other pollutants have smaller effects. Diesel vehicles cause more damages per mile than do gasoline vehicles, because of greater particulate emissions. Very fine particles appear more dangerous than larger particles, and combustion particles appear more dangerous than road dust. The possibility cannot be ruled out that ozone is linked to mortality and chronic illness, effects which are costly and would considerably raise the costs of ozone pollution. These results suggest that emphasis should be placed on the regulation of particulates.

## 1. Introduction

Emissions from motor vehicles and related sources, such as petroleum refineries, have a variety of effects on human health. The effects can be as innocuous as itchy eyes, or as serious as chronic lung disease or heart failure. These physical effects also have an implicit monetary value, which can be useful information to transport analysts and policy makers who wish to perform social cost-benefit analyses of transport investments, prioritise efforts to mitigate damages, or simply examine cost trends.

In this paper, we summarise our recent analysis of the dollar cost of the health effects of motor-vehicle-related air pollution.[1] Although there are many studies of the economic cost of the health impacts of pollution from all anthropogenic sources (see, for example, Krupnick and Portney, 1991; Hall *et al.*, 1992), only two recent studies focus on the health costs of motor vehicle emissions specifically. Small and Kazimi (1995) estimate the health cost of particulate matter and ozone attributable to motor vehicles, but in Los Angeles only. The recent analysis of Krupnick *et al.* (1996) is national in scope, but considers only emissions from refineries and particulate matter emissions attributable to motor vehicles, and probably underestimates emissions related to motor-vehicle use. To our knowledge, no recent analysis examines the cost of all the health effects of all pollutants from all emissions sources related to motor-vehicle use.

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[1] Described in detail in McCubbin and Delucchi (1996).

## 2. The Model

We model the relationship between changes in emissions related to motor-vehicle use and changes in health costs in several steps:
(1) estimate emissions related to motor-vehicle use;
(2) estimate changes in exposure to air pollution;
(3) relate changes in air-pollution exposure to changes in physical health effects;
(4) relate changes in physical health effects to changes in economic welfare.

### 2.1 Emissions related to motor-vehicle use

The production, distribution and combustion of gasoline and diesel introduces hundreds of harmful compounds to the air, land, and water. Our analysis, however, is limited to four of six "criteria" pollutants[2] for which we have air-quality data and dose-response functions: carbon monoxide (CO); nitrogen dioxide ($NO_2$); ozone ($O_3$); and particulate matter (PM), including PM less than 2.5 microns in aerodynamic diameter ($PM_{2.5}$), and PM between 2.5 microns and 10 microns (coarse $PM_{10}$). In addition, we include six "toxic" pollutants linked to cancer: acetaldehyde; benzene; 1,3-butadiene; formaldehyde; gasoline particulates; and diesel particulates.

We do not include sulphur dioxide ($SO_2$) because we have not found enough evidence that $SO_2$ has an effect independent of other pollutants. Although Charpin *et al.* (1988) reported that $SO_2$ might be associated with the incidence of cough, wheezing, runny nose, and eye irritation, and Schwartz (1992) reported that $SO_2$ was linked to the duration of chest discomfort, most studies that examined respiratory ailments and hospital admissions and controlled for more than one air pollutant either could not determine which pollutant was the causative agent (for example, Lipfert and Hammerstrom, 1992), or else could not find an effect for $SO_2$ (for example, Abbey *et al.*, 1993; Hoek *et al.*, 1993; Krupnick *et al.*, 1990; Schwartz *et al.*, 1991; Schwartz and Zeger, 1990).

Similarly, most mortality studies have failed to find a significant link between mortality and $SO_2$, independent of the effect of particulates. Although studies by Moolgavkar *et al.* (1995) and Xu *et al.* (1994) controlled for TSP and found $SO_2$ significantly linked to mortality, several other studies that controlled for TSP (Loomis *et al.*, 1996; Schwartz, 1991; Schwartz and Dockery, 1992a,b), or for other particulate measures such as British Smoke and light reflectivity (Kinney and Ozkaynak, 1991; Mazumdar *et al.*, 1982; Schwartz and Marcus, 1990), failed to find a significant effect for $SO_2$. Samet *et al.* (1997) were unable to distinguish the effect of particulates from the effect of $SO_2$. Moreover, for two reasons, we do not think that TSP is an especially good measure of particulates for an epidemiological study. First, TSP includes larger particulates (greater than 10 micrometers), which are considered less dangerous than smaller particulates. Second, larger particulates have a wide geographic variation that makes a given meas-

[2] Criteria pollutants are those pollutants for which the US Environmental Protection Agency has established national ambient air quality standards.

urement representative of only a small area. Both factors suggest TSP is a poor control variable. We did not find evidence of a significant effect for $SO_2$ when fine particulates was used as a control variable (for example, Dockery *et al.*, 1992).

We do not include lead because it has been phased out of gasoline in the US market. (See Salkever,1995, and Schwartz,1994, for a discussion of the health and economic costs of lead.)

We consider four kinds of sources of emissions related to motor-vehicle use:
(a) tailpipe and evaporative emissions from the vehicles themselves;
(b) emissions from "upstream" fuel and vehicle production processes;
(c) particulate dust emissions from paved roads; and
(d) particulate dust emissions from unpaved roads.

We estimate health effects and present cost results cumulatively: for direct vehicle emissions only (a); for direct vehicle emissions plus upstream emissions (a + b); for direct vehicle emissions plus road-dust emissions (a + b + c), and for all motor-vehicle-related emissions (a + b + c + d). This cumulative analysis (as opposed to an analysis of each source category one at a time) is necessary because the ambient concentrations and the effects of air pollution on health are nonlinear functions of the levels of emissions.

Our estimates of emissions are based on, but not always the same as, the official emissions inventories produced by the United States Environmental Protection Agency.[3] For example, it is likely that the EPA overestimates emissions of particulate road dust, but underestimates emissions of particulate matter from vehicles. In cases such as those, we adjusted the official estimate to what our independent analysis suggested was a more accurate figure. (Appendix A discusses the two most important adjustments, to the estimates of PM tailpipe emissions and PM road-dust emissions.)

## 2.2 Exposure to air pollution

There are two distinct methods to estimate exposure to pollution: one based on modelled exposure in "micro environments", and the other based directly on ambient air-quality data from the EPA's national air-quality monitoring network. We use the micro-environment method to estimate exposure to toxic air pollutants, and ambient air-quality data to estimate exposure to the criteria air pollutants.

*Criteria air pollutants: exposure to ambient air pollution*

We estimate exposure to criteria air pollutants ($O_3$, $NO_2$, CO, and PM), on the basis of ambient (outdoor) air quality readings and population in US counties in 1990 (EPA, 1993a). Given the actual ambient pollutant concentration, we then estimate what the concentration would have been had motor-vehicle-related emissions been reduced by 10 and 100 per cent, using a simple model of emissions, dispersion, and atmospheric chemistry, developed in Delucchi and McCubbin (1996). We consider two levels of

[3] E. H. Pechan Associates (1995) produced a complete anthropogenic emissions inventory for the EPA. The biogenic emissions are from the EPA (1995).

motor-vehicle emissions reductions because the formation of ambient pollution from emissions, and the effect of ambient pollution on people's health, are nonlinear processes in our model. We also include a case where we reduce all anthropogenic emissions by 100 per cent, to give a feel for the relative importance of motor vehicles.

We assume that the ratio of the actual pollution level before and after the change in emissions is equal to the ratio of the modelled pollution level before and after the change. We can then solve for the actual pollution level after the change in emissions:

$$\text{Assume } PP/PI = PP^*/PI^* \qquad (1)$$
$$PP = PI \cdot PP^*/PI^*$$

where:

$PP$ = the estimated actual pollutant level after the change in pollution (eliminate all anthropogenic pollutant-precursor emissions, or eliminate 10 or 100 per cent of motor-vehicle-related pollutant-precursor emissions);

$PI$ = the actual ambient pollutant level in 1990 (data from air-quality monitors; EPA, 1993a; see McCubbin and Delucchi, 1996, for details); and

$PP^*/PI^*$ = the modelled ratio of pollutant levels after the change in emissions to pollutant levels, given the baseline emissions (see Delucchi and McCubbin, 1996, for details of the model).

Our model has several major simplifying assumptions. First, it is most important to understand that we do not estimate the absolute air quality given the baseline emissions or the change in emissions, but rather estimate directly the percentage change in air quality itself. That is, in equation (1), we estimate the *ratio* $PP^*/PI^*$; we do not estimate $PI^*$ and $PP^*$ individually in units of concentration ($\mu g/m^3$). We do this because we have only a crude model of pollutant dispersion and formation, which is not at all suited to estimating absolute concentrations, but which is serviceable for estimating the contribution of one pollution source (such as motor vehicles) *relative* to another source (such as power plants). Also, to estimate the ratio $PP^*/PI^*$ we need to know only the relative contribution to ambient pollution of the different emission sources.

Because we cannot run chemically and physically detailed oxidant and aerosol models for every region of the country, we have chosen instead to estimate the ratio $PP^*/PI^*$ on the basis of a simple model of precursor dispersion and pollutant formation. In essence, our model "apportions" the known (measured) pollutant concentration ($PI$) back to individual emissions sources on the basis of "dispersion-adjusted" emissions of precursors from those sources, where the dispersion adjustments account for differences in location (sources further away from the point of pollutant measurement contribute less), emissions height, and other factors.

Second, we assume that in each county, $c$, the ambient pollution measured at the air-quality monitors is a function of emissions from all the counties in the same Air Quality Control Region (AQCR) as county $c$. We distinguish between emissions generated within county $c$, and emissions generated in all other counties within the same AQCR as $c$,

but we ignore the transport of pollution from one AQCR to another.[4] Of course, we recognise that for some pollutants in some areas, such as ozone in the Northeastern US, long-range transport is important, and ideally should not be ignored. However, we were unable to perform sophisticated inter-regional modelling for the entire US.

Third, we assume that emissions of precursor pollutants (for example, VOCs and $NO_x$ as precursors to ozone, and VOCs, $NO_x$, and $SO_x$ as precursors to PM) disperse as such from the source to the receptor (the ambient air-quality monitor), and then at the receptor participate in the chemical reactions that result in the measured ambient pollutant (ozone or PM). We ignore meteorology and topography and assume that the ambient pollutant is a function only of the precursor emissions at the site of the monitor.

*The formation of ozone*

Ozone is not emitted as such by motor vehicles or any other source, but forms in the atmosphere from a series of photochemical reactions that involve $NO_x$, VOCs, and other compounds. The reaction rate and equilibrium depend in a complex way on the relative abundance of the reactants, temperature, atmospheric mixing, and other factors. The most accurate way to estimate the contribution of each precursor or set of precursors to ozone is to run photochemical grid models, with and without the precursor emissions from specific sources, and estimate the change in the ozone level. Obviously this is very costly to do for the entire US. Instead, we follow Merz *et al.* (1972) and assume that ozone formation is a simple nonlinear function of $NO_x$ and VOC emissions:

$$Ozone = (VOCs)^A (NO_x)^B, \tag{2}$$

where the parameters *VOC*s and $NO_x$ are the quantity of emissions *not* as released, but as modelled to have dispersed from the point of release to the point of exposure (the air quality monitor). The *VOC*s are weighted by their potential to form ozone.

In equation (2), the exponent $A$ determines the sensitivity of ozone to changes in VOC levels, and the exponent $B$ determines the sensitivity of ozone to changes in $NO_x$ levels. We picked values for $A$ (0.55) and $B$ (0.40) so that the resulting ozone sensitivities (defined formally as the percentage change in *Ozone* divided by the percentage change in *VOC* or $NO_x$) were reasonably consistent with the ozone sensitivities we derived from the results of sophisticated ozone air-quality models (see Delucchi and McCubbin, 1996).[5] However, because we estimate the *ratio PP*/PI**, and not *PI** and *PP** individually in units of concentration ($\mu g/m^3$), and because of the nonlinear relationship

[4] We model every county $c$ as a circle covering the centre of a circular AQCR. At the centre of county $c$ and the AQCR is an air-quality monitor, $R$. Upwind of $R$, but within the circular area of county $c$, are that county's motor-vehicle and other emission sources, located at different assumed distances $r$ from the centre monitor(s) $R$. Outside the circle of county $c$, but within the same AQCR, are all the emission sources in all the other counties, assumed to be located at a radial distance $r_0$ from the centre $R$. The distances $r$ and $r_0$, and all the other parameters in the air-quality model, are discussed in Delucchi and McCubbin (1996). Air quality control regions are defined in the Code of Federal Regulations (Section 40: Part 81).

[5] More recent work by Georgopoulos *et al.* (1997) indicates that the ozone VOC sensitivity can range from 0.1 to 0.6, and the ozone $NO_x$ sensitivity from –0.5 to 0.9. (A negative sensitivity means that an increase in $NO_x$ actually decreases ozone levels — a phenomenon known to occur under certain conditions.)

between emissions of precursors and ambient pollutant levels, there is no certain way for us to validate our estimates more completely.

*The formation of secondary particulate matter*

The reactions that form secondary particulates also are complex and difficult to model. To simplify, we assume that 15 to 25 per cent (western US) or 25 to 35 per cent (eastern US) of the $SO_x$ emitted becomes sulphate, that 5 to 7 per cent of the $NO_x$ emitted becomes nitrate, and that the resultant sulphates and nitrates react with ammonia ($NH_3$) to form secondary particulate sulphate and secondary particulate nitrate. We assume that, within an air basin, the $NH_3$ neutralises the sulphate first, to form ammonium bisulphate. If there is more than enough $NH_3$ to convert all the sulphate to ammonium bisulphate, then we assume that the excess $NH_3$ further neutralises the bisulphate to ammonium sulphate. If there is more than enough $NH_3$ to fully neutralise the ammonium bisulphate to ammonium sulphate, then we assume that the $NH_3$ begins to neutralise nitrates. We also include secondary organic aerosols, estimated as a fraction of VOC emissions.

Our model only begins to account for the complexity of ammonium sulphate and nitrate formation. We ignore the effects of weather, relative concentrations, and emission rates, and we consider as precursors only $SO_2$, $NO_x$, and $NH_3$; we do not consider the involvement of dust, water vapour, or other compounds in these reactions.

*A comparison of our PM model results with other estimates of ambient PM pollution*

Because of the relatively high damages attributable to particulate matter, it is important to compare our model of PM air quality with the results of other ways of estimating the contribution of various sources to ambient PM air pollution. One such alternative method is to examine the chemical composition of particulate matter captured at air-quality monitors, and then relate the chemical profile of different emissions sources to the chemical profile of the ambient pollutant. This statistical "chemical mass-balance" (CMB) relationship results in weights, or source-apportionments, for the different emission sources. These CMB source apportionments are analogous to the pollutant shares — $PP^*/PI^*$, from equation (1) — calculated by our model.

Delucchi and McCubbin (1996) present CMB results for 21 counties, mostly in the western US, and 10 sources of particulate matter: primary geologic; primary construction; primary motor vehicle; primary vegetative burning; secondary ammonium sulphate; secondary ammonium nitrate; and four miscellaneous categories. For three out of four source categories — road dust/geologic, secondary ammonium sulphate, and secondary ammonium nitrate — our model results and the CMB results agree reasonably well, although the CMB results are generally more variable.

However, the CMB studies estimate a much larger direct contribution from motor vehicles than our model does. Our model estimates that direct, primary PM emissions from motor vehicles contribute 1 to 9 per cent of ambient PM; the CMB studies estimate that motor vehicles contribute about three times as much, although again there is considerable variability in the CMB results.

We can explain at least some of this difference. For example, the CMB estimates of motor-vehicle emissions probably include PM from non-motor-vehicle diesel combus-

tion. Another explanation of the difference is that CMB studies often sample at times and places of especially high motor-vehicle contributions. Even so, the difference between our model results and the CMB results is conspicuous, and we certainly cannot rule out the possibility that our model is significantly underestimating the direct contribution of motor vehicles to ambient PM, either because it underestimates motor-vehicle emissions or overestimates the contribution of other emission sources.

*Toxic air pollutants: exposure in micro-environments*

With the micro-environment method, people's movements through different "micro-environments" (for example, in a car, or in an office) are modelled over the course of a day. First, pollution levels in each micro-environment are estimated, then the amount of pollution ingested (sometimes taking into account body weight and respiration rate) is estimated, and finally, dose-response functions derived from clinical studies are used to estimate the health effects of the exposure. Examples of such exposure modelling include the Regional Human Exposure (REHEX) model of Hall *et al.* (1989, 1992), the EPA's (1993b) *Hazardous Air Pollutant Exposure Model for Mobile Sources* (HAPEM-MS), and the work of Krupnick and Kopp (1988).

We use the results of one of these micro-environment models, HAPEM-MS (EPA, 1993b), to estimate the effect of toxic air pollution on the incidence of cancer. HAPEM-MS accounts for people's movement each day between micro-environments of varying pollution levels. In this model the average exposure to pollution is the sum of the weighted daily micro-environment exposures, wherein the weights are proportional to the pollutant concentration and the length of time spent in each micro-environment. The EPA (1993b) uses this model to estimate cancer cases caused by cars. We use the EPA's work and a companion work by Johnson *et al.* (1992) to estimate the annual average per capita exposure to acetaldehyde, benzene, 1,3-butadiene, formaldehyde, diesel particulates, and gasoline particulates.

## 2.3 Health effects of exposure

A review of the epidemiological literature suggests that air pollution causes a variety of effects including eye irritation, headaches, acute and chronic respiratory illness, and death. To estimate the health effects of the criteria pollutants, we construct exposure-response functions, typically using the results of logistic and Poisson regression analyses. To estimate the health effects of the toxic air pollutants, we use cancer unit risk estimates.

We reviewed hundreds of clinical, animal, and epidemiological studies of the health effects of various pollutants, and constructed exposure-response functions for each criterion pollutant (ozone, carbon monoxide, and so on), and each of a variety of health effects (for example, asthma or headaches). These functions relate the change in health effects to the change in exposure. For most pollutants and health effects, we have established upper- and lower-bound estimates of the effects of exposure. We consider each of the pollutants in turn.

*Particulate matter*

Particulate matter is the most dangerous pollutant and perhaps the most complicated. It is a heterogeneous mix of solid or liquid compounds, including organic aerosols, sulphates, nitrates, and metals, suspended in the atmosphere. It comprises a very wide range of chemical constituents, structures, and sizes, which in turn can have a wide range of physiological and biological effects. Because the size distribution and chemical composition of PM vary from one emissions source to the next (for example, particulate matter from diesel engines is substantially different from particulate matter from road dust), it is likely that some emissions sources contribute more to the estimated overall harm than do others. To attribute the estimated health effects to motor vehicles correctly, we must specify the properties of particulate matter that are most harmful.

Unfortunately, the epidemiological studies that we use tend to relate health effects to an undifferentiated mass of particles, without distinguishing chemical compounds or several size classes.[6] Thus, we must decide which kinds and sizes of particles are the most harmful.

Current research indicates that smaller particles are more dangerous than larger particles (Dockery *et al.*, 1995; Marrack, 1995; Pope *et al.*, 1995). When we apportion total particulate damages to individual sources (such as motor vehicles), we assume that $PM_{2.5}$ is from 2.0 (lower bound) to 10.0 (upper bound) times as potent (in terms of damages per gram) as is the coarse fraction of $PM_{10}$. We assume that particulates larger than 10 microns, which generally are not deeply inhaled, are not harmful at all.

It is possible that very fine particles ($PM_1$, or even smaller) cause more damage than do the particles in the $PM_1$ to $PM_{2.5}$ range. Ideally, one would know the continuous size distribution of particles from each emissions source, and the damages as a continuous function of size. However, there are simply not enough data to establish such functions.

Regarding the chemical composition of particulates, the evidence suggests that fugitive dust particulates from paved and unpaved roads, road construction, and agricultural tillage are less harmful than other types of particulates, but not entirely harmless (Ziegler *et al.*, 1994; Hefflin *et al.*, 1994; Kleinman *et al.*, 1995; Koenig *et al.*, 1995). Our best estimate is that dust *of a given size class* could be anywhere from about half as potent as other (mainly combustion) particles, to an order of magnitude less potent. One could certainly make different assumptions.

*Mortality associated with particulate pollution*

Death is, of course, the most serious health effect, and particulates appear to be the most deadly air pollutant. The epidemiological evidence suggests that there are three types of deaths that we need to distinguish when we value mortality: "acute harvest" deaths; "acute non-harvest" deaths; and "chronic" deaths.

Acute deaths are those that occur only a relatively short time — say, less than a week — after exposure to particulate air pollution. There are two kinds of acute deaths due to pollution: harvest deaths and non-harvest deaths. Acute harvest deaths are those that

[6] Some studies (Ozkaynak and Thurston, 1987; Dockery *et al.*, 1992; Pope *et al.*, 1996) did include sulphate, nitrate, or acidic particulates, but the results of these studies are inconclusive.

would have occurred in a few days anyway even if there had there been no particulate pollution. We distinguish between harvest and non-harvest deaths because the former cost only a few days or weeks of life, whereas the latter cost years, and we believe that the value of the death is related at least crudely to the number of days or years lost.

Chronic deaths are those that occur many years after the precipitating exposure to particulate air pollution. If exposure to air pollution initiates a cancer or cardiopulmonary disease that years later is fatal, the resulting death is called a "chronic" death. We distinguish between acute and chronic because the latter occur much later than do the former and hence have a lower present value, all else being equal.

The available epidemiological studies do not estimate separately acute harvest deaths, acute non-harvest deaths, and chronic deaths. Instead we have three kinds of epidemiological studies: time-series, prospective cohort, and cross-sectional studies.[7] Time-series studies (see, for example, Pope *et al.*, 1992) link daily mortality with ambient particulate concentration. They estimate total acute deaths, both harvest and non-harvest, but do not necessarily distinguish between acute harvest and acute non-harvest deaths. Generally they cannot capture chronic deaths. Prospective cohort studies (such as Pope *et al.*, 1995) keep track of individuals over many years and link observed mortality with long-term average pollution levels. They are designed to control for important confounding factors such as smoking and are able to estimate acute non-harvest deaths and chronic deaths, but do not distinguish between the two. Prospective cohort studies may capture harvest deaths; but we assume that they do not. Cross-sectional studies (see, for example, Ozkaynak and Thurston, 1987), also called "ecological" regressions, link (retrospectively) annual average pollution level with annual mortality rates in metropolitan regions. Although these regressions are common, so too is the criticism that they do not adequately control for important confounding variables, such as smoking, except by using city-wide averages.[8] Like prospective cohort studies, they estimate acute non-harvest deaths and chronic deaths. They do not capture acute harvest deaths.

To estimate acute harvest deaths, we deduct from total time-series deaths an estimate of the number of acute non-harvest deaths therein. To estimate chronic deaths, we deduct from total prospective cohort deaths (or cross-sectional deaths[9]) an estimate of the number of acute non-harvest deaths therein. We estimate acute non-harvest deaths as some fraction of total time-series or total prospective cohort deaths. It appears to us that there is little ground for assuming that in the time-series studies, pollution is *mainly* harvesting deaths. We assume that acute harvest deaths are 25 per cent (upper-bound cost case) to 50 per cent (lower-bound cost case) of total time-series-estimated acute deaths. Acute non-harvest deaths are therefore are 75 to 50 per cent. We subtract these estimated acute non-harvest deaths from total cross-sectional deaths, to obtain chronic deaths.

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[7] Lipfert and Wyzga (1995a) give a useful summary of different types of mortality studies.

[8] Criticisms of cross-sectional studies are considered in Evans *et al.* (1984) and Lipfert and Wyzga (1995a,b), among others.

[9] To estimate non-harvest acute deaths and chronic deaths, we use a cross-sectional study (Ozkaynak and Thurston, 1987) in the lower bound, and a prospective cohort study (Pope *et al.*, 1995) in the upper bound.

*Chronic illness associated with particulate pollution*

Chronic illness is perhaps the most difficult health effect to quantify. Chronic diseases may develop very gradually, after many years of exposure, and hence not be associated with any single year or episode of exposure. Moreover, more than one factor might contribute to the development of chronic illnesses. To isolate the contribution of air pollution to chronic illness, a study must cover many years, carefully monitor exposure to pollution, and control for all contributory factors.

A number of studies have found that particulates cause chronic respiratory problems in people of all ages. Chapman *et al.* (1985), in a study of young adults, and Dockery *et al.* (1989) and Vedal *et al.* (1991), in studies of grade school children, found particulates linked to chronic cough, chronic phlegm, wheezing, chest illness, and bronchitis. In a series of studies of adult Seventh Day Adventists living in California, Abbey *et al.* (1993; 1995) found TSP and $PM_{10}$ significantly linked to chronic respiratory illness. We use the work of Abbey *et al.* (1995) to develop a dose-response function for chronic illness and particulate air pollution, because they gathered data for over ten years and controlled for other health risks by choosing a population (Seventh-Day Adventists) that smokes and drinks little.

*Carbon monoxide*

Carbon monoxide is dangerous because it binds with haemoglobin in the blood to form carboxyhaemoglobin, thereby reducing the oxygen carrying capacity of the blood and limiting the release of oxygen from circulating haemoglobin. CO especially affects individuals with heart trouble (EPA, 1991, pp.2-17), and is linked to headaches (Schwartz and Zeger, 1990). Three recent studies (Schwartz, 1997; Morris *et al.*, 1995; Schwartz and Morris, 1995) provide reasonably compelling evidence of a link between CO and cardiovascular problems.[10] Morris *et al.* (1995) and Schwartz and Morris (1995) found CO linked to hospital admissions for congestive heart failure, and Schwartz (1997) found CO linked to cardiovascular hospital admissions. Both the Schwartz and Morris (1995) and Schwartz (1997) studies controlled for $PM_{10}$ — an important control variable.

Regarding mortality, the available evidence suggests that CO exposure increases the risk of death for some people.[11] However, nobody has quantified this risk convincingly and in a way that we can use to establish a dose-response function that directly links CO with mortality (that is, apart from the indirect link with mortality via the link between CO and hospital admissions for heart failure).[12]

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[10] Several older studies (Edling and Axelson, 1984; Kurt *et al.*, 1978; Stern *et al.*, 1981, Stern *et al.*, 1988) provided evidence that CO increases cardiovascular problems, but, unfortunately, they did not adequately control for other pollutants.

[11] The EPA (1991, pp.1-12) concluded that the evidence is "suggestive but not conclusive" that CO may lead to sudden death in people with coronary artery disease.

[12] We did include an indirect effect of CO on mortality by using the work of Morris *et al.* (1995) and assuming that 6 per cent of the estimated cases of hospital admissions for congestive heart failure result in mortality.

*Nitrogen dioxide*

A number of laboratory and epidemiological studies suggest that $NO_2$ increases minor respiratory symptoms and eye irritation. We used Schwartz and Zeger's (1990) work to estimate the increase in the number of days with sore throat, excess phlegm, and eye irritation. We did not find evidence linking $NO_2$ to more serious illnesses such as emphysema, bronchitis, and cancer, nor did we find satisfactory evidence of a link between $NO_2$ and mortality.

*Ozone*

Ozone is a strong oxidant linked with a number of adverse health effects, including eye irritation[13] (Schwartz and Zeger, 1990), asthma attacks (Holguin *et al.*, 1985; Whittemore and Korn, 1980), and other acute lower and upper respiratory symptoms (Krupnick *et al.*, 1990; Ostro *et al.*, 1993; Ostro and Rothschild, 1989; Portney and Mullahy, 1986). Although scientists have linked ozone to acute respiratory problems, they have not yet found strong evidence that ozone increases the number of people suffering from chronic illness. The EPA (1996, pp.7-171) concluded that chronic health effects studies to date have failed to prove such a link exists, but that "the aggregate evidence to date suggests that chronic ozone exposure, along with other environmental factors, could be responsible for health effects in exposed populations." We do not include any relationship between ozone and chronic illness.

At the time of our analysis, the evidence regarding an independent link between ozone and mortality was mixed, and one might reasonably have doubted that ozone had any effect on mortality apart from the effect of other pollutants. Kinney and Ozkaynak (1991), Moolgavkar *et al.* (1995), and Kinney *et al.* (1995) did find a significant mortality effect for ozone, but Kinney and Ozkaynak, and Moolgavkar *et al.*, used relatively poor measures of particulates — KM (similar to British smoke) and TSP — and Kinney *et al.* found that the effect of ozone became insignificant in models with $PM_{10}$ included. Thus, when we carried out our analysis we recognised that there could be an independent link between ozone and mortality, but believed that it was more likely that such a link would double count the mortality effects of other pollutants. However, more recent studies have strengthened the link between ozone and mortality. In a careful re-analysis of air pollution in Philadelphia, with TSP, CO, $SO_2$, $NO_2$, and ozone as independent variables, Samet *et al.* (1997, p.25) found "a strong independent effect of $O_3$ ...consistent across seasons and across different models for the other pollutants." A recent meta-analysis (EPA, 1997a, Appendix J) suggests that ozone may indeed have an effect independent of particulates and other pollutants, although the effect is an order of magnitude smaller than that of particulates. On the other hand, Loomis *et al.* (1996) found that in

[13] Lippman (1989) noted that oxidants other than ozone, such as peroxyacetyl nitrate and aldehydes, are responsible for increased eye irritation. However, given that these other oxidants usually occur together with ozone, and that the contribution of motor vehicles to ambient levels of these other oxidants is about the same as the contribution to ambient ozone (30 to 50 per cent), we may use ozone as a proxy to estimate the effect of motor vehicles on eye irritation.

Mexico City neither $SO_2$ nor ozone had an effect on mortality that was statistically independent of particulate pollutant levels, and as a result they "could not resolve whether high ambient ozone levels would have an important effect on mortality when particulate levels were reduced" (p.25). If, on the basis of some of these recent studies, we were to include an independent mortality effect of ozone, the estimated ozone damages might increase considerably.

*Cancer risk from toxic air pollutants*

Motor vehicles emit a wide range of toxic compounds, which can be acutely or chronically poisonous, carcinogenic, teratogenic, or mutagenic. However, the available emissions, exposure, and health data allow us to model only one type of effect (cancer) of only six toxic air pollutants (acetaldehyde, 1,3-butadiene, benzene, diesel particulates, formaldehyde, and gasoline particulates).

To translate exposure into estimated cancer cases, we use the linear cancer model — also termed the "one-hit" model because even very small amounts of toxics cause cancer — which requires the estimation of a unit-risk number.[14] A unit-risk number estimates the excess risk of cancer, to one individual, from 70 years (a lifetime) of continuous exposure to one microgram per meter cubed ($\mu g/m^3$).

To estimate the number of excess cancers to a population from exposure to a toxic air pollutant in a given year, we determine the number of "exposure-years" at an exposure level of one $\mu g/m^3$. (For example, the exposure of seventy people to one $\mu g/m^3$ for a year is equal to 70 exposure-years.) Multiplying the number of exposure-years by the unit-risk number then tells the number of cancer cases that would arise from exposure to motor vehicles.

Because the social cost of cancer depends very much on whether or not the victim recovers, and the rate of recovery differs from one type of cancer to the next, we specify the type of cancer caused by each toxic air pollutant. To do this, we use the epidemiological and laboratory results cited by the EPA (1993b). For example, benzene is linked to an increased incidence of leukemia in studies of workers in rubber factories; consequently, we assume that benzene from motor vehicles causes leukemia.[15] We estimate the recovery rate for each type of cancer from statistics provided by the American Cancer Institute.

It is well known that the development and diagnosis of cancer does not occur concurrently with the emission of toxics. There is a lag or latency period, which we assume may extend anywhere from one to fifty years. The longer the lag between emission and health effect, the lower the present value of the cost of the emissions. We assume that, on average, cancer is discovered 25 years after exposure, and that the incidence of cases follows, roughly, a normal distribution.

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[14] The EPA (1993b) gives more details on the evidence linking particular toxics to cancer, and the development of unit-risk numbers (both the EPA's own estimates and estimates by others).

[15] The other toxic-cancer linkages are based on animal studies. We assume that if toxic A causes cancer X in animals, it causes cancer X in humans as well.

## 2.4 Valuing health effects

In our final step, valuing health effects, we merge the results from the epidemiological literature, which links air pollution to illness, with the results from the economic literature, which places a value on illness. There are a number of health effects that we value: acute morbidity, chronic morbidity, mortality and cancer. Ideally we would perfectly match the effect that we are estimating with the same effect from the economic literature and ensure that we appropriately value mild, moderate, and severe effects. However, this is impossible to do, and the result is that we do not value the health effects that we estimate as precisely as we would like. This is the "matching problem" discussed by Krupnick (1988, pp.2-6).

There are other issues. People may not value reducing the *N*th day of illness as much as the first, either because of income constraints, or simply because of declining marginal utility of "consumption" of health benefits. Reviewing the available literature, Hall *et al.* (1989, pp.5-47) proposed a declining, nonlinear relationship between the average willingness to pay (WTP) to avoid many symptom days, and the WTP to avoid one symptom day. Although there are problems with their formulation, it gives plausible results, and we use it in our own valuation.

Similarly, it is possible that the WTP to avoid many different symptoms simultaneously is not equal to the sum of the WTP to avoid the symptoms one at a time. Because we do not estimate simultaneous multi-symptom illnesses, we do not account for this.

*Acute morbidity*

Economists use several methods to estimate the cost of such things as the common cold, asthma and other illnesses, which we group under the category of acute morbidity. In their review article Cropper and Freeman (1991, p.166) summarised the two general approaches. The "observed market" approach includes "techniques that rely on demand and cost functions, market prices, and observed behaviour and choices." Household production functions and cost-of-illness studies (which estimate the direct out-of-pocket expenses of illness) are examples. The "constructed market" approach includes techniques that directly ask people's "willingness to pay or accept compensation for a postulated change, how their behaviour would change, or how they would rank alternative situations involving different combinations of health and income or consumption." Contingent valuation is an example.

The advantage of the observed-market approach is that it is based on actual behaviour; the disadvantage is that the markets being observed value only part of what we wish to value, or else value it only implicitly, as part of a bundle of goods. The constructed-market approach avoids this problem by specifying precisely and explicitly what is to be valued; it is reliable only insofar as people respond realistically to the constructed market.

As a result, the available valuation estimates are uncertain and often unreliable. We agree with Krupnick and Kopp (1988, p.2-59), who concluded that the imprecision of the available estimates of morbidity costs preclude a definitive point-estimate of the cost. We select a wide range of values from the literature (Table 1).

**Table 1**

*Differences in Health Values between the Lower- and Upper-Bound Scenarios*

| | *Pollutant* | *Lower Bound* | *Upper Bound* |
|---|---|---|---|
| **Acute morbidity**[a] | | | |
| Headache | CO | $3 | $14 |
| Sore throat | $NO_2$ | $3 | £14 |
| Excess phlegm | $NO_2$ | $3 | $14 |
| Eye irritation | $NO_2$, $O_3$ | $3 | $14 |
| Respiratory illness excluding asthma | $O_3$ | $3 | $14 |
| Any other symptom (ARD2) | $O_3$ | $3 | $14 |
| Asthma attack | $O_3$, PM | $10 | $50 |
| Respiratory restricted activity day (RRAD) | PM | $20 | $70 |
| **Chronic illness**[b] | PM | $0.5 million | $2 million |
| **Value of mortality** | | | |
| Cancer from toxics[c] | toxics | $0.5 million | $2 million |
| Harvest death[d] | PM | $10,000 | $50,000 |
| Non-harvest death[e] | PM | $1 million | $4 million |
| Present value of chronic death[f] | PM | $0.513 million | $3.8 million |
| **Value of life: rate of increase per year**[g] | - | 1% | 1.5% |
| **Discount rate** | - | 8% | 2% |

a To estimate the cost of acute morbidity, we used judgemental values based in large part on the work of Krupnick (1988, chapter 6), Krupnick and Kopp (1988), Hall *et al.* (1989), and Krupnick and Portney (1991).

b The cost of chronic illness is based on Viscusi *et al.* (1991) and Krupnick and Cropper (1992).

c When valuing cancer, we assume that people would be willing to pay at least as much to avoid cancer as they would pay to avoid chronic illness.

d The value of a harvest death is based on our best judgement.

e Starting with a range of $2 million to $5 million per lost life, we used lower values to account for the fact that a significant portion of the deaths that we are estimating are of the elderly or of individuals with compromised health.

f The average present value of a chronic death is based on the assumption that deaths occur over a twenty-year period (following a roughly normal distribution).

g The rate of increase in the value of life is used when valuing chronic deaths, which occur a number of years after exposure to pollution. We assume that the value of life is related to real wealth and that value of life in the future can be calculated based on annual increase in wealth. As discussed in McCubbin and Delucchi (1996, pp.145-48), we assume that the value of life will increase 1.0 to 1.5 per cent annually.

*Chronic illness*

Particulates cause asthma, chronic bronchitis and emphysema, which we refer to as a group as airway obstructive disease. Krupnick and Cropper (1992) reported that people are willing to pay $0.46 million to $2.08 million to avoid a statistical case of chronic bronchitis, and Viscusi *et al.* (1991) reported $0.46 million to $2.29 million per statistical case of chronic bronchitis. We assume values of $0.5 million and $2.0 million in our lower and upper bound.

*Mortality*

As discussed above, we estimate three different kinds of mortality: acute harvest deaths, acute non-harvest deaths, and chronic deaths. Here, we estimate different values of life (VOL) for each.

(1) We assume that the acute harvest deaths estimated from the time-series studies have forgone only a few days or weeks of life, which we value at $10,000 to $50,000.

(2) The acute non-harvest deaths estimated from the overlap of time-series and cross-sectional studies have forgone much more life, and consequently should be valued much more. But how much living is forgone, and at what value? Our review of the literature suggests that the VOL for a middle-aged person is in the range of $2 million to $5 million. However, air pollution appears to kill mainly the elderly. A wage-risk study by Moore and Viscusi (1988) indicates that the VOL of a 70-year old with a life expectancy of 75 years is 40 to 46 per cent of the VOL of a 40-year old. A contingent valuation study by Jones-Lee *et al.* (1985) indicates that a 70-year-old values her life at about 71 per cent of the value of a 40-year-old. Both sets of results suggest that it is appropriate to reduce the value of the lives of the elderly. Therefore, we assume that the VOL here ranges from $1 million to $4 million.

(3) We assume that *chronic deaths* will occur over a twenty-year period. We value them at $1 million to $4 million at the time they occur, and then discount the value of these future deaths with an 8 per cent and a 2 per cent discount rate, in the lower bound and upper bounds, to obtain the present value.

*Cancer*

Toxic pollutants such as benzene and diesel particulates increase the risk that people develop cancer. Cancers often have a long latency period, and are expensive and time-consuming to treat. The chance of recovery depends on many factors, including the age of the person and the type of cancer.

For *fatal* cancer cases, we assign the value of a statistical life at the point at which the cancer is discovered, and ignore, on the one hand, costs incurred between the time of discovery and death, and, on the other, the time between discovery and death. The omission of post-discovery costs understates the present value of the cost of cancer, but the failure to consider the time lag between discovery and death overstates the present value (since we use a positive discount rate), so that these two simplifications tend to cancel.

On the basis of an estimate by Rae *et al.* (1991), and estimates of the cost of chronic disease, we choose $0.5 million as a lower-bound estimate of the cost of non-fatal cancer, and $2 million for an upper-bound estimate.

## 3. Results

Tables 2 and 3 present the damages caused by all anthropogenic air pollution and by motor-vehicle-related air pollution. The most striking results are the large damages caused by ambient particulate matter (PM), and the large contribution of motor vehicles to ambient particulate levels. Ambient CO, ozone, $NO_2$, and toxics all cause much smaller damages than does ambient PM. It is especially interesting to note that the damages from ambient ozone, which is the most heavily regulated criteria pollutant, are less than the damages from CO and $NO_2$, and fully two orders of magnitude less than the damages from ambient PM (Tables 2 and 3). The damages from toxic air pollutants appear to be of the same order of magnitude as the ozone damages (Table 3). PM damages are so high because PM pollution kills or chronically sickens far more people than do the other pollutants, which typically cause relatively mild and far less costly effects, such as headaches and mild respiratory distress.[16]

For any particular ambient pollutant, the upstream emissions sources contribute only a minor part of the total motor-vehicle-related damage (Table 3). The damages from road dust have a wide range, on account of uncertainty regarding the emissions and potency of road dust, but could be quite large.

Table 4 shows the cost per vehicle mile travelled for the nation, for urban areas, and for the Los Angeles metropolitan area. Again, PM pollution has a very large, albeit uncertain cost. As we would expect, the cost per mile is highest in Los Angeles, which has high levels of pollution and a high population density, and is lowest over the whole nation, which includes some rural areas with low pollution levels and few people. Table 4 also shows that diesel vehicles have a higher cost than gasoline powered vehicles, mainly because diesels emit more particulates.

Apart from their contribution to particulate formation, emissions of nitrogen dioxide, sulphur dioxide, and volatile organic carbon, are relatively unimportant.

Table 5 shows the cost per kilogram of pollutant emitted. Direct emissions of PM have the highest cost/kg, followed by emissions of $NO_x$ and $SO_x$ Emissions of CO are the least costly per kg. Note, though, that emissions of $NO_x$ are far more costly as precursors to secondary particulate matter than they are as precursors to Ozone or to direct $NO_2$ formation. Note too that the cost per kilogram of tailpipe emissions is highest, and the cost per kg of tailpipe + road dust + upstream the lowest. This is because upstream sources are more remote, and road dust less potent, than are tailpipe emissions.

We caution that these results apply to 1990 emission levels. The Clean Air Act Amendments of 1990 mandated significant reductions in emissions from many sources, and as a result, vehicles and upstream emissions sources in the future will emit less per mile of travel and have a lower $/VMT cost than they did in 1990. To estimate the $/VMT cost of future, cleaner vehicles and upstream emissions sources, multiply the per-

[16] It is always possible, too, that the effects that we attribute to PM should be attributed (at least partly) to other pollutants or to something other than air pollution. Several recent papers (Moolgavkar and Luebeck, 1996; Moolgavkar *et al.*, 1995; Lipfert and Wyzga, 1995a,b; Ito *et al.*, 1993) argue that it is too early to conclude that particulates are the prime cause of pollution-related damages.

**Table 2**
*National (USA) Cases of Adverse Health Effects and their Cost due to Anthopogenic Pollution in 1990*

| *Emission* | *Ambient Pollutant* | *Health Effect* | *Thousands of Cases* | | *Cost of Cases (1991 $ billion)* | |
|---|---|---|---|---|---|---|
| | | | *Low* | *High* | *Low* | *High* |
| CO | CO | Headache | 170,385 | 202,416 | 0.4 | 2.5 |
| | | Hospitalisation | 8 | 23 | 0.1 | 0.2 |
| | | Mortality | 0.5 | 1.5 | 0.5 | 5.5 |
| | | All | - | - | 1.0 | 8.2 |
| $NO_x$ | $NO_2$ | Sore throat | 265,577 | 269,583 | 0.6 | 3.0 |
| | | Excess phlegm | 121,800 | 123,700 | 0.4 | 1.7 |
| | | Eye irritation | 109,618 | 111,303 | 0.3 | 1.5 |
| | | All | - | - | 1.3 | 6.1 |
| VOC + $NO_x$ | $O_3$ | Asthma attacks | 3,652 | 11,482 | 0.04 | 0.5 |
| | | Eye irritation | 33,852 | 37,383 | 0.1 | 0.6 |
| | | Lower respiratory illness | 48,584 | 81,089 | 0.2 | 1.2 |
| | | Upper respiratory illness | 14,782 | 24,672 | 0.05 | 0.4 |
| | | ARD2 | 0 | 276,144 | 0.0 | 3.2 |
| | | All | - | - | 0.3 | 5.8 |
| $PM_{10}$, $SO_2$, $NO_x$, VOC | $PM_{10}$[a] | Asthma attacks | 3,003 | 3,172 | 0.03 | 0.2 |
| | | RRAD | 88,673 | 120,133 | 2.0 | 8.2 |
| | | Chronic illness | 39 | 93 | 10.0 | 173.8 |
| | | Mortality | 80 | 137 | 40.1 | 470.0 |
| | | All | - | - | 52.1 | 652.1 |
| All | All | All | - | - | 54.7 | 672.3 |

a Includes particulate sulphates, particulate nitrates, and organic particulates, as well as direct or primary particulate emissions.

tinent $/kg factors of Table 5 by the associated estimated future kg/mile emission rates. The $/kg factors are independent of the kg/mile emission rate, and so can be applied to any future emission rate. They are, however, proportional to the *exposed* population; such that if the exposed population rises by X% then the $/kg factor should be scaled up by X%.

**Table 3**
*Cost of Motor Vehicle Air Pollution,*
*Based on a 100 per cent Reduction in Motor-Vehicle-Related Emissions*
*(1991 $ million)*

| *Ambient Pollutant* | *Motor Vehicles* | | *Motor Vehicles + upstream* | | *Motor Vehicles + upstream + road dust*[a] | |
|---|---|---|---|---|---|---|
| | *Low* | *High* | *Low* | *High* | *Low* | *High* |
| **Nation** | | | | | | |
| $PM_{10}$ | 16,727 | 266,391 | 18,961 | 279,354 | 21,943 | 432,829 |
| $O_3$ | 214 | 1,899 | 228 | 1,945 | 228 | 1,945 |
| $NO_2$ | 1,038 | 5,483 | 1,048 | 5,509 | 1,048 | 5,509 |
| CO | 919 | 8,085 | 921 | 8,092 | 921 | 8,092 |
| Toxics | 87 | 1,622 | 87 | 1,623 | 87 | 1,623 |
| Total | 18,985 | 283,481 | 21,246 | 296,522 | 24,227 | 449,997 |
| **Urban** | | | | | | |
| $PM_{10}$ | 15,954 | 253,126 | 18,059 | 265,228 | 20,599 | 401,037 |
| $O_3$ | 196 | 1,730 | 209 | 1,771 | 209 | 1,771 |
| $NO_2$ | 955 | 5,072 | 964 | 5,093 | 964 | 5,093 |
| CO | 829 | 7,089 | 831 | 7,094 | 831 | 7,094 |
| Toxics | 76 | 1,411 | 76 | 1,412 | 76 | 1,412 |
| Total | 18,010 | 268,428 | 20,139 | 280,599 | 22,679 | 416,408 |
| **Los Angeles CMSA**[b] | | | | | | |
| $PM_{10}$ | 4,203 | 51,869 | 4,291 | 52,564 | 4,633 | 76,260 |
| $O_3$ | 33 | 265 | 34 | 266 | 34 | 266 |
| $NO_2$ | 137 | 713 | 137 | 714 | 137 | 714 |
| CO | 99 | 746 | 100 | 746 | 100 | 746 |
| Toxics | 3 | 63 | 3 | 63 | 3 | 63 |
| Total | 4,476 | 53,656 | 4,565 | 54,353 | 4,968 | 79,033 |

[a] Includes both paved and unpaved road dust.
[b] The Los Angeles Consolidated Metropolitan Statistical Area (CMSA) consists of Los Angeles, Riverside, Orange, San Bernadino, and Ventura counties.

### 3.1 Comparison with studies of air pollution costs in the South Coast Air Basin

We can compare our results with those of Krupnick and Portney (1991), Hall *et al.* (1992), and Small and Kazimi (1995), who estimated the health cost of ozone and particulate pollution in the South Coast Air Quality Management District. In Table 6, we compare their estimates with our estimates for the Los Angeles region.

## Table 4

*Cost per Mile of Motor Vehicle Travel, based on a 10 per cent Reduction in Motor-Vehicle-Related Emissions*
*(cents per vehicle mile travelled in the USA in 1990)*

| *Vehicle Type* | *Emission Source*[a] | *PM* | | *$O_3$* | | *CO* | | *$NO_2$* | | *Toxics* | | *Total* | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Low* | *High* | *Low* | *High* | *Low* | *High* | *Low* | *High* | *Low* | *High* | *Low* | *High* |
| LDGV | *v* | 0.48 | 7.02 | 0.01 | 0.07 | 0.04 | 0.37 | 0.04 | 0.22 | 0.00 | 0.05 | 0.58 | 7.71 |
| | *v+u* | 0.56 | 7.50 | 0.01 | 0.07 | 0.04 | 0.37 | 0.04 | 0.22 | 0.00 | 0.05 | 0.66 | 8.20 |
| LDGT | *v* | 0.74 | 10.70 | 0.01 | 0.11 | 0.06 | 0.53 | 0.06 | 0.32 | 0.00 | 0.09 | 0.88 | 11.72 |
| | *v+u* | 0.90 | 11.54 | 0.01 | 0.11 | 0.06 | 0.53 | 0.06 | 0.32 | 0.00 | 0.09 | 1.04 | 12.56 |
| HDGV | *v* | 1.56 | 30.28 | 0.03 | 0.28 | 0.15 | 1.63 | 0.12 | 0.76 | 0.01 | 0.29 | 1.86 | 33.12 |
| | *v+u* | 1.78 | 31.53 | 0.03 | 0.29 | 0.15 | 1.63 | 0.12 | 0.77 | 0.01 | 0.29 | 2.09 | 34.38 |
| Gasoline | *v* | 0.55 | 8.04 | 0.01 | 0.08 | 0.05 | 0.42 | 0.05 | 0.25 | 0.00 | 0.06 | 0.65 | 8.83 |
| | *v+u* | 0.64 | 8.61 | 0.01 | 0.08 | 0.05 | 0.42 | 0.05 | 0.25 | 0.00 | 0.06 | 0.75 | 9.40 |
| LDDV | *v* | 1.47 | 18.49 | 0.00 | 0.02 | 0.00 | 0.01 | 0.02 | 0.11 | 0.01 | 0.08 | 1.50 | 18.64 |
| | *v+u* | 1.50 | 18.70 | 0.00 | 0.02 | 0.00 | 0.01 | 0.02 | 0.11 | 0.01 | 0.08 | 1.53 | 18.84 |
| LDDT | *v* | 0.47 | 5.77 | 0.00 | 0.01 | 0.00 | 0.00 | 0.01 | 0.04 | 0.00 | 0.03 | 0.48 | 5.82 |
| | *v+u* | 0.52 | 6.14 | 0.00 | 0.01 | 0.00 | 0.00 | 0.01 | 0.04 | 0.00 | 0.03 | 0.53 | 6.19 |
| HDDV | *v* | 4.18 | 79.93 | 0.02 | 0.19 | 0.01 | 0.07 | 0.15 | 0.98 | 0.02 | 0.33 | 4.35 | 81.19 |
| | *v+u* | 4.43 | 81.37 | 0.02 | 0.20 | 0.01 | 0.07 | 0.15 | 0.99 | 0.02 | 0.33 | 4.61 | 82.63 |
| Diesel | *v* | 3.48 | 64.86 | 0.01 | 0.15 | 0.00 | 0.05 | 0.12 | 0.78 | 0.01 | 0.27 | 3.62 | 65.85 |
| | *v+u* | 3.68 | 66.03 | 0.02 | 0.16 | 0.01 | 0.05 | 0.12 | 0.78 | 0.01 | 0.27 | 3.83 | 67.03 |
| All | *v* | 0.78 | 12.57 | 0.01 | 0.09 | 0.04 | 0.39 | 0.05 | 0.29 | 0.00 | 0.08 | 0.89 | 13.37 |
| | *v+u* | 0.89 | 13.17 | 0.01 | 0.09 | 0.04 | 0.39 | 0.05 | 0.29 | 0.00 | 0.08 | 1.00 | 13.98 |

*PM*

| | *v* | | *v+u* | | *v+u+rd* | | *v+u+rd+re* | |
|---|---|---|---|---|---|---|---|---|
| | *Low* | *High* | *Low* | *High* | *Low* | *High* | *Low* | *High* |
| LDGV | 0.48 | 7.02 | 0.56 | 7.50 | 0.60 | 10.90 | 0.65 | 12.18 |
| LDGT | 0.74 | 10.70 | 0.90 | 11.54 | 0.94 | 16.09 | 1.02 | 17.80 |
| HDGV | 1.56 | 30.28 | 1.78 | 31.53 | 1.90 | 42.55 | 2.07 | 46.69 |
| Gasoline | 0.55 | 8.04 | 0.64 | 8.61 | 0.68 | 12.33 | 0.74 | 13.74 |
| LDDV | 1.47 | 18.49 | 1.50 | 18.70 | 1.53 | 21.34 | 1.57 | 22.34 |
| LDDT | 0.47 | 5.77 | 0.52 | 6.14 | 0.57 | 10.09 | 0.63 | 11.58 |
| HDDV | 4.18 | 79.93 | 4.43 | 81.37 | 4.75 | 110.91 | 5.21 | 122.01 |
| Diesel | 3.48 | 64.86 | 3.68 | 66.03 | 3.93 | 89.55 | 4.30 | 98.39 |
| All | 0.78 | 12.57 | 0.89 | 13.17 | 0.94 | 18.47 | 1.02 | 20.46 |

[a] Each emission source is cumulative: *v* includes just motor vehicle emissions; *v+u* includes *v* plus upstream emissions; *v+u+rd* includes *v+u* plus paved road dust emissions; *v+u+rd+re* includes *v+u+rd* plus unpaved road dust emissions.

**Table 5**

*Cost per Kilogram of Motor Vehicle Emissions in the USA in 1990*

*(1991 $)*

| *Source*[a] | *Emission* | *Ambient Pollutant* | *United States* | | *Urban Areas* | | *Los Angeles* | |
|---|---|---|---|---|---|---|---|---|
| | | | *Low* | *High* | *Low* | *High* | *Low* | *High* |
| *v* | CO | CO | 0.01 | 0.09 | 0.01 | 0.10 | 0.03 | 0.18 |
| | $NO_x$ | nitrate-$PM_{10}$ | 1.02 | 16.56 | 1.39 | 22.38 | 6.05 | 75.83 |
| | | $NO_2$ | 0.15 | 0.73 | 0.19 | 0.96 | 0.52 | 2.64 |
| | Total for $NO_x$ | | 1.17 | 17.29 | 1.59 | 23.34 | 6.58 | 78.47 |
| | $PM_{2.5}$ | $PM_{2.5}$ | 10.42 | 159.19 | 14.81 | 225.36 | 63.98 | 779.13 |
| | coarse $PM_{10}$ | coarse $PM_{10}$ | 6.70 | 17.68 | 9.09 | 23.89 | 38.12 | 78.34 |
| | Total for $PM_{10}$ | | 9.75 | 133.78 | 13.74 | 187.47 | 58.79 | 638.33 |
| | $SO_x$ | sulphate-$PM_{10}$ | 6.90 | 65.52 | 9.62 | 90.94 | 34.98 | 226.89 |
| | VOC | organic-$PM_{10}$ | 0.10 | 1.15 | 0.13 | 1.45 | 0.51 | 4.34 |
| | VOC + $NO_x$ | ozone | 0.01 | 0.11 | 0.02 | 0.14 | 0.05 | 0.40 |
| *v+u* | CO | CO | 0.01 | 0.09 | 0.01 | 0.10 | 0.03 | 0.18 |
| | $NO_x$ | nitrate-$PM_{10}$ | 0.96 | 15.53 | 1.31 | 21.17 | 6.02 | 75.11 |
| | | $NO_2$ | 0.14 | 0.68 | 0.18 | 0.91 | 0.52 | 2.62 |
| | Total for $NO_x$ | | 1.10 | 16.21 | 1.50 | 22.08 | 6.54 | 77.73 |
| | $PM_{2.5}$ | $PM_{2.5}$ | 9.71 | 147.24 | 13.63 | 205.44 | 62.57 | 737.36 |
| | coarse $PM_{10}$ | coarse $PM_{10}$ | 5.30 | 14.25 | 7.20 | 18.34 | 30.04 | 47.87 |
| | Total for $PM_{10}$ | | 8.78 | 116.01 | 12.23 | 158.23 | 54.68 | 509.18 |
| | $SO_x$ | sulphate-$PM_{10}$ | 2.80 | 22.60 | 4.40 | 35.28 | 33.53 | 209.88 |
| | VOC | organic-$PM_{10}$ | 0.10 | 0.99 | 0.13 | 1.25 | 0.52 | 4.29 |
| | VOC + $NO_x$ | ozone | 0.01 | 0.10 | 0.02 | 0.12 | 0.05 | 0.39 |
| *v+u+rd* | $PM_{2.5}$ | $PM_{2.5}$ | 7.48 | 94.45 | 10.47 | 130.89 | 45.44 | 430.10 |
| | coarse $PM_{10}$ | coarse $PM_{10}$ | 1.03 | 7.58 | 1.42 | 10.34 | 5.62 | 32.26 |
| | Total for $PM_{10}$ | | 2.84 | 39.87 | 3.92 | 54.64 | 15.20 | 169.45 |
| *v+u+rd+re* | $PM_{2.5}$ | $PM_{2.5}$ | 3.22 | 45.22 | 6.53 | 88.79 | 41.93 | 405.29 |
| | coarse $PM_{10}$ | coarse $PM_{10}$ | 0.27 | 2.95 | 0.63 | 6.20 | 4.69 | 29.77 |
| | Total for $PM_{10}$ | | 0.60 | 15.13 | 1.45 | 31.69 | 12.35 | 155.58 |

[a] Each emission source is cumulative: *v* includes just motor vehicle emissions; *v+u* includes *v* plus upstream emissions; *v+u+rd* includes *v+u* plus paved road dust emissions; *v+u+rd+re* includes *v+u+rd* plus unpaved road dust emissions.

## Table 6

*Air Pollution Damages in the Los Angeles Region*

*(1992 $ millions)*

| | *This study*[a] | | *Hall et al. (1992)*[b] | | *Krupnick & Portney (1991)*[b] | *Small & Kazimi (1995)* |
|---|---|---|---|---|---|---|
| | *All anthropogenic emissions in Los Angeles region*[c] *in 1990* | | *Emissions reductions required to achieve air quality standards in SCAQMD in 2010* | | | *All emissions including biogenic in SCAQMD in 1990* |
| *Damage* | *Low* | *High* | *Low* | *High* | *Low* | *High* |
| **Particulates** | | | | | | |
| Mortality | 5,093 | 48,062 | 3,439 | 17,668 | 2,372 | 30,180[d] |
| Morbidity | 2,727 | 38,924 | 919 | 919 | 830 | [e] |
| **Ozone** | | | | | | |
| Morbidity | 52 | 655 | 1,423 | 6,878 | 356 | [e] |
| Total | 7,872 | 88,642 | 5,781 | 25,465 | 3,557 | [e] |

Note: SCAQMD = South Coast Air Quality Management District (Orange County, and portions of Los Angeles, Riverside, and San Bernadino counties).

[a] Our original 1991 $ estimates are scaled by the 1991/92 CPI (1.03), for comparison with the 1992 $ estimates of Small and Kazimi (1995).

[b] Hall *et al.* (1992) and Krupnick and Portney (1991) report their results in 1988 $. We have scaled their results to 1992 $, for comparison with the results of Small and Kazimi (1995). In their own comparison, Small and Kazimi (1995) use the 1992/1988 CPI (1.186, in their analysis) to scale the results of Hall *et al.* (1989) and Krupnick and Portney (1991). We do the same.

c All of Los Angeles, Orange, San Bernadino, and Riverside counties.

d We have extrapolated from Small and Kazimi's damage estimate for motor vehicle air pollution emissions to an estimate of damages caused by all anthropogenic air pollution emissions. This extrapolation is based on the geometric mean (0.893) of the mortality coefficients from Evans *et al.* (1984) (imputed $PM_{10}$ coefficient = 0.615) and Ozkaynak and Thurston (1987) (imputed $PM_{10}$ coefficient = 1.298), an annual average $PM_{10}$ concentration of 57.8μg/m$^3$, a population of 12 million, and a value of life of $4.87 million. (See Small and Kazimi, 1995, p.18 and footnote 11.)

Note that Small and Kazimi underestimate the lower bound coefficient (from Evans *et al.*) when they noted (p.18): "Based on the consensus that $PM_{10}$ rather than TSP causes health damages, we assume that among the constituents of TSP, only $PM_{10}$ causes mortality; then the same coefficient (0.338) applies to changes in $PM_{10}$." Actually, they needed to correct for the $PM_{10}$ fraction of TSP, which we assume is 55%, as noted by Hall *et al.* (1989, pp.4-20). The coefficient should be 0.615 (= 0.338/0.55).

Finally, note that anthropogenic sources contibute roughly 90% of ambient particulates. To make this estimate more comparable to our own, one should subtract 10% of their estimate to account for natural emissions.

e Small and Kazimi (1995, p.20) used the results of Hall *et al.* (1992) and Krupnick and Portney (1991) to estimate morbidity costs of ozone and particulates. We did not attempt to extrapolate from Small and Kazimi's motor vehicle results to estimate the morbidity costs of all anthropogenic ozone and particulates.

**Table 7**
*Two Estimates of the Cost of Ozone and Particulate Air Pollution from Motor Vehicles in the Los Angeles Region*
*(cents per vehicle-mile travelled)*

| | *This study (PM and $O_3$ damages only)*[a] | | | | *Small & Kazimi (1995)*[b] |
|---|---|---|---|---|---|
| | *Direct Emissions*[c] | | *Direct + Upstream + Road Dust* | | *Direct Emissions* |
| *Vehicle* | *Low* | *High* | *Low* | *High* | *Baseline* |
| Light-duty Gasoline | 2.1 | 23.2 | 2.4 | 36.5 | 3.3 |
| Light-duty Diesel | 6.7 | 64.4 | 6.9 | 74.7 | 7.8 |
| Heavy-duty Diesel | 20.5 | 324.0 | 22.9 | 462.8 | 52.7 |

[a] We have converted our estimates from 1991 $ to 1992 $ by multiplying by the 1992/91 CPI of 1.03. The estimates of Small and Kazimi (1995) are in 1992 dollars.
[b] Small and Kazimi's (1995) estimates apply to the SCAQMD, described in Table 6. Our estimates apply to the Los Angeles Consolidated Metropolitan Statistical Area (CMSA), described in Table 3. Note that because here we compare cents per VMT, and not total dollar damages, the difference between the SCAQMD and the Los Angeles CMSA is relatively unimportant.
[c] Direct emissions include the formation of secondary particulates from $NO_x$, $SO_2$, $NH_3$, and VOCs.

**Table 8**
*The Contribution of Motor Vehicles to Total Damages from Particulate Air Pollution in the South Coast Air Basin*

| | *The fraction of total ambient PM from motor-vehicle-related emissions of:* | | | | | |
|---|---|---|---|---|---|---|
| *County* | *Direct PM* | | *Precursors of secondary ambient PM* | | | *Total* |
| | *Coarse $PM_{10}$* | *$PM_{2.5}$* | *$NO_x$* | *$SO_x$* | *Organic Compounds* | |
| | % | % | % | % | % | % |
| Our upper bound[a] | | | | | | |
| Los Angeles | 0.5 | 19.2 | 30.5 | 5.8 | 1.9 | 57.8 |
| Orange | 0.4 | 16.4 | 27.3 | 5.0 | 1.7 | 51.0 |
| Riverside | 0.3 | 14.5 | 21.9 | 4.3 | 1.3 | 42.2 |
| San Bernadino | 0.3 | 13.5 | 21.1 | 4.0 | 1.2 | 40.3 |
| Our lower bound[a] | | | | | | |
| Los Angeles | 2.1 | 13.7 | 25.5 | 10.4 | 2.2 | 53.8 |
| Orange | 1.9 | 12.7 | 23.6 | 9.6 | 2.1 | 50.0 |
| Riverside | 1.6 | 11.5 | 19.9 | 8.4 | 1.7 | 43.1 |
| San Bernadino | 1.5 | 10.5 | 18.6 | 7.7 | 1.6 | 39.9 |
| Small & Kazimi (1995)[b] | 10.6[c] | | 10.5 | 5.3 | 4.4 | 30.8 |

[a] The percentages shown are modelled shares of ambient PM. The emissions and air-quality model used to estimate these shares is discussed in Delucchi and McCubbin (1996).
[b] Small and Kazimi (1995) calculate the share of motor-vehicle tailpipe emissions. They do not weight PM emissions by potency.
[c] Small and Kazimi (1995) report the share of $PM_{10}$, which is coarse $PM_{10}$ plus $PM_{2.5}$.

The four estimates differ widely, mainly on account of differences in particulate mortality and morbidity costs. Our estimates of mortality costs are higher than those of Krupnick and Portney (1991) and Hall *et al.* (1992), because we estimate a larger number of deaths, and, in comparison with Krupnick and Portney, a higher value of life. Hall *et al.* and Krupnick and Portney reported that reducing pollution levels to the ambient air quality standard would save 1,600 to 2,000 lives in the South Coast Air Quality Management District; we estimate that removing anthropogenic particulate pollution would save 9,700 to 13,100 lives in Los Angeles. Our estimate comes closest to Small and Kazimi's because they estimate that 4,000 to 9,000 lives are lost annually, and use a slightly higher value of life than we do.

There are two main reasons why our estimate of deaths is higher than the previous studies.

(1) We estimate the effects of all anthropogenic pollution, whereas Krupnick and Portney and Hall *et al.* estimate the effects of the reductions necessary to achieve ambient air quality standards in the year 2010. We estimate the effects of all anthropogenic pollution because it appears that there are health effects at concentrations below the ambient air quality standard.

(2) We include deaths estimated from both time-series studies and cross-sectional studies, whereas Hall *et al.* (1992), Krupnick and Portney (1991), and Small and Kazimi (1995) used only cross-sectional studies. Most importantly, in our upper bound case we use a recent prospective cohort study (Pope *et al.*, 1995) that estimates more "cross-sectional" deaths per unit change in PM pollution.

We estimate a higher cost of morbidity due to particulates, primarily because we include an estimate of the cost of chronic illness, and the others do not. Our estimate of the cost of ozone morbidity is lower than the estimate of Hall *et al.* (1992) because they use laboratory studies to estimate acute morbidity damages, and, in general, laboratory results give higher estimates than epidemiological studies.

### 3.2 Small and Kazimi's study: The cost of motor-vehicle air pollution in Los Angeles

Small and Kazimi (1995) estimated the cost of motor-vehicle particulate and ozone air pollution in the South Coast Air Quality Management District. We can compare their estimates with our estimates of the cost of motor-vehicle ozone and particulate air pollution in Los Angeles.

Small and Kazimi (1995) estimated the contribution to ambient $PM_{10}$ levels of *direct* PM emissions from motor vehicles, and the contribution to secondary PM of VOC, $NO_x$, and $SO_x$ emissions from motor vehicles, and then used a dose-response function derived from Ozkaynak and Thurston (1987) and Evans *et al.* (1984) to estimate the health impacts of the motor-vehicle PM pollution. They used the results of Krupnick and Portney (1991) and Hall *et al.* (1992) to estimate morbidity due to ozone and particulate. Their resulting cost estimates for the 1992 fleet are shown in Table 7. The results shown in Table 7 do not include the cost of upstream emissions, which they did not estimate,

or the cost of road dust, which they estimated but excluded from their baseline results on account of the uncertainty in the road-dust emissions inventory. They calculated that dust from paved roads would have added 4.3 cents per mile to the cost of light--duty vehicles. This would have resulted in a total cost of 7.6 cents per mile for light-duty gasoline vehicles, compared to 2.4 to 36.5 cents per mile in our analysis.

The estimates of Small and Kazimi (1995) fall within our ranges, but at the low end. There are two reasons why our upper-bound estimates are so much larger than Small and Kazimi's baseline estimates. First, as shown in Table 7, our upper-bound estimate of the total cost of all anthropogenic ozone and particulate pollution in Los Angeles is larger than Small and Kazimi's estimate, mainly because we include more effects than they do: we include chronic illness from particulates and time-series as well as cross-sectional deaths from particulates. Second, in our upper bound (and on average) we attribute a larger fraction of the total ozone and particulate damages to motor vehicles. Small and Kazimi apportion particulate damages on the basis of the contribution of particulates sources to the downtown Los Angeles monitoring station, and apportion ozone damages on the basis of the emission shares of ozone precursors. By contrast, we use a Gaussian plume dispersion model, and simple models of the formation of ozone and secondary particulate matter, to estimate the contribution of motor vehicle emissions to ambient pollution in each county in the Los Angeles region. The resulting contribution of motor vehicle tailpipe emissions to total PM are shown in Table 8.

## 4. Discussion

Most regulations and analyses have focused on emissions of ozone precursors from motor vehicles. Yet, in our lower-bound case, ozone is the least damaging pollutant emitted directly from motor vehicles — less damaging even than CO and $NO_x$ — and in fact is an order of magnitude less damaging than road dust, which no one has even considered regulating. Although we found relatively minor effects for ozone, recent evidence suggests that ozone (in addition to particulates) may be implicated with mortality; if so, this would significantly raise our estimate of ozone's costs.

Although there is considerable uncertainty in our analysis, it is clear that, over a wide range of assumptions, damages from particulates dominate the total cost of the health effects of motor-vehicle air pollution. We suggest that regulatory and analytical efforts be broadened and redirected towards examining particulates. Certainly, we find no basis for the presumption, too common among many analysts, that the carbon-monoxide problem can now be considered to be "solved" (even if CO levels come down by a factor of two, CO damages still will be of the same order of magnitude as ozone damages), or that fugitive dust can be ignored.

The difference between our lower- and upper-bound estimates of particulate damages is considerable — typically a factor of ten to twenty. This great uncertainty, while unsettling, properly reflects the current gaps in our understanding of the links between emissions, exposure, health effects, and economic value.

For many pollutants, the emission inventory is uncertain, primarily because the underlying emission factors (in grams/unit of activity) are not well characterised. Our model of air quality and exposure is simplistic, but it probably does not have a relatively large source of uncertainty, because it is unlikely that the actual contribution of motor vehicles to ambient ozone differs from our modelled contribution by more than 50 per cent. This potential error is fairly small compared to the ranges of uncertainties in valuing damages.

Most studies have assumed that the health effects associated with a particular pollutant depend on that pollutant only, and not on interactions between pollutants. Recent studies suggest that a suite of pollutants may interact among themselves and with environmental variables (such as pollen, weather) to cause adverse effects (Moolgavkar and Luebeck, 1996; Moolgavkar *et al.*, 1995; Lipfert and Wyzga, 1995a,b; Lebowitz *et al.*, 1992). More research with more sophisticated models is needed to address this question.

Air pollution probably has different effects on different age groups. These differences, which have not been well quantified, could be important because society may wish to attach different values to damages to different age groups. Future work should determine damage coefficients by age group.

Particulates cause most of the estimated damages, and are the source of the greatest uncertainty in the analysis. Several important questions still need to be answered.

(1) Do prospective cohort studies capture the same acute deaths that time-series studies capture? (We assume that they capture the same non-harvest deaths, and we subtract those, estimated by time-series studies, from deaths estimated by a prospective hohort study, to estimate chronic deaths.) What fraction of acute deaths are harvest deaths, and how well do prospective cohort studies capture these?

(2) What is the correct mortality coefficient — the change in the death rate per unit change in particulate pollution? Two recent, prospective cohort studies (Dockery *et al.*, 1993; Pope *et al.*, 1995), which are probably the best studies available, have relatively large mortality coefficients, two to four times larger than those from previous studies.

(3) What is the effect of particulate air pollution on chronic illness? Although recent work indicates that air pollution harms the growth, development, and health of the respiratory system, it is difficult to quantify this effect in a way that is useful for cost analyses.

(4) To what extent does the effect of PM depend on the size of the particle? Many researchers believe that "fine" particulate matter (that with a diameter of 2.5 microns or less) is more dangerous than larger particulate matter, but the functional relationship between particle size and health effects has not been quantified.

(5) To what extent does the effect of PM depend on the chemical composition of the particles? There is some indication that road dust and other soil- and mineral-based particulate matter is less damaging than sulphate particulate matter from combustion, but the evidence is only suggestive, and certainly does not yet demonstrate that mineral-based particulates such as road dust have no adverse health effects at all.

All the values that we use to quantify air pollution damages in monetary terms are uncertain. The difference between the lower and upper bounds varies by a factor of roughly two to four.[17] Certainly more work would be helpful to refine our estimates further, particularly with regard to our estimates of morbidity and the value of life. With regard to the latter, there are at least two issues.

(1) Are the available estimates of the value of life (actually, value of risk) pertinent to the risks posed by air pollution specifically?
(2) Is the value of life a function of the age of the individual, or the number of years of living lost?

Although further research will help to answer these and other questions, and reduce the uncertainty in the valuation step, it is unlikely that society will ever agree on point estimates for damage values, and that we will always have to choose plausible lower and upper bounds.

## Appendix A

### Adjustments to EPA Estimates of Road Dust and Tailpipe Particulate Emissions

The EPA uses an emission factor model, *PART5*, to estimate tailpipe, tyre-wear, and road-dust emissions of particulate matter from motor vehicles. In this appendix, we briefly review *PART5*, explain why we think it under-estimates tailpipe emissions and overestimates road-dust emissions, and describe the adjustments we made to the EPA emissions inventory to account for these suspected biases.

The *PART5* model, which is a companion to EPA's *MOBILE5* model, calculates gram-per-mile exhaust emissions of PM and $SO_x$, and tyre-wear and road-dust PM emissions, from gasoline and diesel light-duty and heavy-duty cars and trucks. (It does not estimate brake-wear emissions.) Because tyre-wear and brake-wear certainly are relatively insignificant, we do not determine the accuracy of the *PART5* emission factors for these sources. The $SO_x$ emission factors can be presumed to be reasonably accurate, because they are calculated on the basis of the sulphur content of the fuel (EPA, 1995a), which is known fairly accurately.

*Tailpipe emissions*

*PART5* estimates of PM exhaust emissions are suspect. We are concerned most about the accuracy of the estimates of PM exhaust emissions from heavy-duty diesel vehicles (HDDVs), because HDDVs emit many times more PM per mile than do light-duty gasoline vehicles. The emission factors in *PART5* appear to be based on emissions results for

[17]We should point out that our upper-bound estimate of the value of the life is much less than some values reported in the literature and assumed in other studies of health effects. Consequently, we could argue that our treatment of uncertainty is conservative, and that the overall gap between the lower and the upper bound damages is even greater than estimated here.

22 in-use heavy-duty diesel engines tested over the heavy-duty transient cycle (HDTC) in 1983 and 1984 (Guensler *et al.*, 1991; Ostria, 1996). The engines were tested "as received", and then tuned up and tested again. Guensler *et al.* (1991) speculate that the *PART5* emission factors are based on the results of the tests conducted *after* the engines were tuned up. Similarly, Walsh (1995) believes that the PM exhaust emissions from diesel vehicles are calculated on the assumption that the vehicles meet the pertinent model-year PM emission standards, with little if any deterioration over time (this can be confirmed by comparing the output of *PART5* with the relevant emissions standards).

Perhaps more seriously, it appears to us that none of the engines tested were high-emitters, because even the highest level measured in the tests (2.14 g/bhp-hr) is much less than one would expect from a badly smoking engine. Given that the small amount of high-emitters that one typically observes in a fleet can significantly raise fleet-average emissions (Durbin *et al.*, 1999), the omission of high-emitting engines from the emissions tests would have resulted in a significant underestimate of real-world emissions. Also, it is not clear if the vehicles tested and the test cycle used were broadly representative of the in-use fleet and real driving conditions (Guensler *et al.* (1991).

It appears, then, the *PART5* PM emission factors are based on emissions from a small number of tuned-up engines driven over a somewhat idealised test cycle. If so, then in the real world, there are more high-emitting vehicles, and more high-emitting driving, than in the drive tests that were used to establish the factors in *PART5*.

The available emissions data, reviewed in Delucchi and McCubbin (1996) do indicate — persuasively in our view — that *PART5* is underestimating exhaust emissions of PM.

First, chassis dynamometer tests of HDDVs indicate that at only 60,000 miles — well below the midpoint of the life of an HDDV— emissions are already at or above the level predicted by *PART5*. This suggests to us that a fleet of HDDVs, which on average has more than 100,000 miles of travel per vehicle, emits more exhaust PM than is predicted by *PART5*.

Second, several recent independent chassis dynamometer tests of high-mileage, high-emitting, in-use light-duty gasoline vehicles have shown that PM exhaust emissions can vary by over two orders of magnitude, and generally are much higher than predicted by *PART5* (Sagebiel *et al.*, 1996; Cadle *et al.*, 1998, 1997a,b; Durbin *et al.*, 1999). As Cadle *et al.* (1997b) note, "it is clear that the current in-use, high-mileage, older vehicles can have significantly higher $PM_{10}$ emission rates than new vehicles, and higher than the rates used in the EPA...model" (p.3408). All these results are consistent with the hypothesis that *PART5* models relatively new, properly functioning vehicles, not a real "in-use" fleet with some old or malfunctioning vehicles. In fact, a comparison of the emission test results reported in Cadle *et al.* (1998) with the predictions of *PART5* indicates that *PART5* underestimates in-use emissions from light-duty gasoline vehicles by at least a factor of two. Cadle *et al.* (1998b) conclude that: "the failure [of *PART5*] to include high emitters will result in a significant underestimation of the light-duty fleet average $PM_{10}$ emission rate" (p.3).

Third, Delucchi and McCubbin (1996) show that the ratio of emissions of road dust to exhaust emissions from highway vehicles, in the EPA's emissions inventory, is many times higher than the ratio of dust to motor-vehicle exhaust at ambient air-quality monitors. If the ambient ratios are accurate, and if the differences between the ambient ratios and the emissions ratios cannot be explained entirely by differences in emissions dispersion (which, it seems, they cannot), then the estimates of road-dust emissions are too high, or the *PART5*-based estimates of highway-vehicle PM emissions are too low, or, most likely, both.

Finally, *EMFAC7F*, the emission-factor model used in California, apparently incorporates a factor meant to account for high emissions from poorly maintained vehicles (Guensler *et al.*, 1991), and estimates PM emission rates about 1.8 times as high as the *PART5* estimates.

The data reviewed above suggest that *PART5* underestimates emissions from real on-road vehicles, primarily because *PART5* seems to be based on low-mileage, properly functioning vehicle, and takes little, if any, account of super-emitters. In our low-cost case, we assume that the *PART5* model underestimates PM exhaust emissions by a factor of 1.5. In our high-cost case, we assume that *PART5* underestimates exhaust emissions by a factor of 2.0.

*Road-dust emissions*

Motor-vehicle traffic kicks up the dust on the road. Some of this "emitted" road dust is small enough to be suspended in the air as particulate matter. Surprisingly, such "re-entrained road dust", as it is called, is one of the largest sources of particulate matter in the official US emissions inventory that we used in our analysis: in 1990, road dust from paved and unpaved roads accounted for nearly half of all $PM_{10}$ emissions in the US emissions inventory (EPA, 1995c).

Because road dust is apparently such a large source of emissions, it is important to determine if the emission-factor equations used to calculate road-dust emissions are accurate. In this section, we present evidence that the current EPA (1995a) emission factors, used in the *PART5* model, substantially overestimate emissions of $PM_{10}$ and especially $PM_{2.5}$ from paved roads.[18]

Delucchi and McCubbin (1996) discuss several possible sources of error in the development and application of the EPA's emission-factor equation for road dust, and conclude that: (a) it is possible that $PM_{2.5}$ was inaccurately or insufficiently measured in the original emissions tests; (b) the road-dust emission-factor equation might apply only to paved roads with a relatively high silt loading; (c) most paved roads might have a lower silt loading than is assumed in the calculation of the emission inventory; and (d) emissions from HDDVs might be a larger part of total road-dust + vehicle emissions than is currently estimated.

[18]We have not come across any evidence that EPA's (1995b) estimate of $PM_{10}$ dust emissions from unpaved roads is seriously in error, and as a result we do not make any corrections to the $PM_{10}$ emissions inventory for unpaved roads. However, CMB studies indicate that AP-42 overestimates the ratio of $PM_{2.5}$ to $PM_{10}$, so we have adjusted the $PM_{2.5}$ estimates to be consistent with the CMB data.

There is in fact compelling evidence that the paved-road-dust emission factors in the EPA's emission-factor handbook (EPA, 1995c), and the EPA emissions inventories that we used that were based on those factors, overestimate emissions of $PM_{10}$ and $PM_{2.5}$ from paved-road dust (see Delucchi and McCubbin, 1996, for details). First, as mentioned above, the ratio of road-dust PM actually measured in ambient air to motor-vehicle-exhaust PM actually measured in ambient air is much less than the ratio of estimated paved-road-dust emissions to estimated motor-vehicle exhaust emissions. Second, it appears that the EPA equation for road-dust emissions substantially over-predicts PM emissions from roads in the eastern and western US. Third, measurements of the size distribution of paved-road-dust particulate matter indicate that the $PM_{2.5}/PM_{10}$ ratio assumed in the emissions inventory that we use is too high.[19]

On the basis of our analyses of source-apportionment studies of ambient $PM_{10}$, measurements of PM emissions from road traffic, and analyses of the size distribution of dust particles from paved roads, we conclude that, in the emission inventory that we use,[20] $PM_{10}$ road dust from paved roads is overestimated by a factor of 1.2 to 3.3, and that $PM_{2.5}$ emissions are overestimated by 1.7 to 14.

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[19] Shortly after it produced the emission inventory that we use, the EPA revised its $PM_{2.5}/PM_{10}$ ratio for road dust downwards. The ratio that the EPA now uses is more consistent with the data, albeit perhaps still a bit on the high side.

[20] In its most recent emissions inventory, the EPA makes two significant changes to its method of estimating road-dust emissions, which reduce estimated $PM_{10}$ emissions by a factor of about 2.7 (compare EPA, 1997b, with EPA, 1995a). Thus, the most recent EPA estimates of road-dust $PM_{10}$ emissions are in line with ours.

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